Absolute Humidity, Temperature, and Influenza Mortality: 30 Years of County-Level Evidence from the United States

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Recent research exploring associations between environmental factors and influenza outcomes has devoted substantial attention to the role of absolute humidity. However, the existing literature provides very little quantitative epidemiologic evidence on the relations between absolute humidity and other weather variables and influenza outcomes in human populations. In the present study, the authors helped fill this gap by analyzing longitudinal weather and influenza mortality data, observed every month between January 1973 and December 2002, for each of 359 urban US counties. A flexible regression model was used to simultaneously explore fully nonlinear relations between absolute humidity and influenza outcomes and temperature and influenza outcomes. Results indicated that absolute humidity was an especially critical determinant of observed human influenza mortality, even after controlling for temperature. There were important nonlinear relations; humidity levels below approximately 6 g of water vapor per kilogram of air were associated with increases in influenza mortality. Model predictions suggested that approximately half of the average seasonal differences in US influenza mortality can be explained by seasonal differences in absolute humidity alone. Temperature modestly influenced influenza mortality as well, although results were less robust.

disease susceptibility; disease transmission, infectious; humidity; influenza, human; mortality; temperature; viruses

Abbreviations: CI, confidence interval; ICD, International Classification of Diseases.

Estimates suggest that influenza causes 1,700–59,000 deaths annually in the United States (1–6). Total morbidity from influenza is poorly understood but also represents a significant public health threat. Despite the widespread prevalence of influenza, mechanisms driving influenza host susceptibility, disease transmission, and virus survival remain controversial (7, 8).

Observers have long suspected that environmental factors are among the key determinants of influenza incidence, primarily because outbreaks exhibit pronounced seasonal and geographic patterns. Early experimental studies found that solar insolation reduces influenza virus survival (9–11). More recent, related investigations showed that increased vitamin D levels may enhance host immunity (12–14). Other work found that low relative humidity and low temperature favor influenza virus survival and disease transmission (15–18). Cold temperatures and precipitation may also cause susceptible persons to move indoors, thereby increasing disease transmission (19, 20).

Of late, the literature exploring environmental determinants of influenza has devoted substantial attention to absolute humidity. In contrast to relative humidity, which is a function of water vapor and temperature, absolute measures of humidity isolate the water vapor content in a mass (or parcel) of air. A reevaluation of an experimental study involving guinea pigs found that low absolute humidity is better than low relative humidity at predicting influenza transmission (21, 22). Recent epidemiologic evidence, using aggregate state-level data from the United States, showed that anomalous drops in absolute humidity predict the onset of human influenza outbreaks, as measured by excess mortality (23). Emerging simulation evidence on
Third, the study observed on larger geographic scales like states or nations. The authors believe that this analysis makes several contributions to the field. First, the data permitted the first large-scale ecologic analysis of the impact of absolute humidity and other weather conditions on human influenza mortality. Second, weather data observed at the county level reduced misclassification errors that may plague data observed on larger geographic scales like states or nations. Third, the study’s statistical methods controlled for numerous possible confounders, reducing the possibility that observed weather-influenza links were driven by omitted factors independently associated with both environmental factors and influenza. Fourth, the analysis considered temperature and humidity simultaneously, allowing the researchers to separately assess: 1) the effects of humidity holding other weather conditions constant and 2) the effects of temperature holding other weather conditions constant. Since temperature and absolute humidity are naturally related (7, 23), models that individually examine temperature or humidity will inaccurately estimate their respective effects on influenza morbidity and mortality.

MATERIALS AND METHODS

Influenza mortality data

Mortality data were obtained from the National Center for Health Statistics’ Multiple Cause of Death files (24). All deaths with an International Classification of Diseases (ICD) code associated with influenza as a primary or secondary cause were categorized as influenza mortality (25, 26). Codes from the Eighth Revision of the ICD (ICD-8) were used for the years 1973–1978, codes from the Ninth Revision (ICD-9) were used for the years 1979–1998, and codes from the Tenth Revision (ICD-10) were used for the years 1999–2002. Dushoff et al. (6) showed that these multiple-cause influenza classifications are significantly more robust than single underlying-cause classifications alone. For each county and month, mortality rates per 100,000 persons were calculated by dividing the total influenza death counts by the estimated population in 100,000s. Population data for each county-year were obtained from the National Cancer Institute’s Surveillance, Epidemiology, and End Results system (27).

It is likely that influenza mortality rates are measured with error. Influenza deaths are not always corroborated virologically, and deaths due to influenza may be attributed to other respiratory and circulatory diseases (6, 28). ICD coding practices changed in 1979 and 1999. ICD coding may vary across seasons, and misclassification may be more prevalent in summer months. ICD coding may also vary systematically across locations because of differences in age profiles, availability of virus surveillance, and other factors.

Weather data

Weather data were obtained from the National Climatic Data Center’s Global Summary of the Day files (29). The weather data are organized by station and day. Daily mean temperatures were reported in degrees Fahrenheit (°F). Daily specific humidity, a common measure of absolute humidity, in grams of water vapor per kilogram of air (g/kg) was calculated from daily dew point and atmospheric pressure data, following standard meteorologic formulas (30, 31). Daily total precipitation was obtained in inches (1 inch = 25.4 mm). All station-days missing data on absolute humidity were dropped (0.4% of all station-days), and all station-days missing data on precipitation were dropped (3.0% of all station-days). Stations missing more than half of the absolute humidity and/or precipitation observations for any given year between 1973 and 2002 were then dropped entirely.

Associations between environmental factors and mortality are probably nonlinear, but the precise functional forms are unknown a priori. Commonly postulated relations for temperature and mortality are U- or J-shaped (32). One recent study presents evidence that the relation between absolute humidity and all-cause mortality is reverse J-shaped (33). Consequently, for each station-day, for the present study we constructed all weather exposure assessments from piecewise splines. Splines allow the relations between weather conditions and mortality to vary flexibly at different points along a factor’s distribution but do not require the researchers to choose a specific functional form ahead of time. For this reason, the use of splines is increasingly common in the epidemiologic literature (34).

In our application, piecewise cubic splines were constructed with the STATA 11 (StataCorp LP, College Station, Texas) command “mkspline,” so that the final relation between mortality and the given weather condition was a piecewise function composed of polynomial segments (35). Note that a cubic spline allows for smoothness at the knots, unlike a linear spline. For the technically inclined reader, our absolute humidity functions had knots at 3, 6, 9, 12, 15, and 18 g/kg. Temperature functions had knots at 15, 30, 45, 60, 75, and 90°F (−9.4, −11.1, 7.2, 15.6, 23.9, and 32.2°C), and precipitation functions had knots at 0.25, 0.50, 0.75, and 1.00 inches (6.4, 12.7, 19.1, and 25.4 mm).

Analysis sample

The final unit of analysis was county by month. No spatial or temporal aggregation was necessary for mortality outcomes, which are reported at the county-month level in the Multiple Causes of Death public-use data set. However, both spatial and temporal aggregation was necessary for the
weather exposure variables, as weather conditions are reported at the station-day level.

Aggregating weather exposure to county-month-level independent variables involves 3 steps. First, the spline exposure variables described above were aggregated to the county-day level by calculating the inverse distance-weighted average over all stations within 50 miles (80 km) of each county’s geographic centroid (36). Second, the county-day exposure variables were averaged across days within a county-month. Note that averaging spline variables preserves weather information from all days, including extreme weather days. Third, the weather exposure variables were averaged over the current and preceding month. This 2-month moving average lag structure allows time for disease transmission, permits lags between infection and death, and permits lags between surveillance culture results and ICD coding. Evidence suggests that such lags may be important (6, 37). An additional advantage of the 2-month moving average is that it minimizes potential biases from short-term “harvesting” (32, 38). If the weather accelerates some deaths by a few days or weeks, these deaths will not affect our estimates. We denote our final exposure functions as \( f(\text{HUMID}), g(\text{TEMP}), \) and \( h(\text{PRCP}) \).

The final longitudinal data set contained 129,240 observations on 359 urban counties observed over a period of 360 months spanning January 1973 to December 2002. The 359 densely populated counties in the sample contained 65% of the total US population over the sample period. More rural counties were not evaluated, for data availability reasons. Counties with fewer than 100,000 inhabitants between 1973 and 2002 lacked complete mortality data and were not evaluated. In addition, 11% of counties with complete mortality data lacked complete weather station data and thus were not evaluated.

**Statistical analysis**

Our primary statistical strategy in this study was to regress a given county’s influenza mortality in a given time period on flexible nonlinear functions of that county’s absolute humidity, temperature, and precipitation in the recent past (a 2-month moving average). The basic statistical model was

\[
\text{MORT} = f(\text{HUMID}) + g(\text{TEMP}) + h(\text{PRCP}) + \mu_c + \Phi_t + \epsilon.
\]

MORT is the influenza mortality rate for a given county and month. \( f(\text{HUMID}), g(\text{TEMP}), \) and \( h(\text{PRCP}) \) are the exposure functions for absolute humidity, temperature, and precipitation. The model estimates several parameters for each exposure function. \( \mu_c \) represent estimated county-by-calendar month fixed-effect parameters, and \( \Phi_t \) are estimated time period fixed-effect parameters. \( \epsilon \) is a normally distributed error term.

The county-by-calendar month fixed effects \( \mu_c \) are included so that our model estimates relations between anomalous mortality rates and anomalous weather conditions for a given county and calendar month. This approach is equivalent to estimating seasonal mortality baselines for each county and then analyzing excess deaths only. These fixed effects also minimize omitted variable concerns that might arise from potential confounders that vary across counties and/or seasons but remain roughly constant across years. Examples of potential cross-county or seasonal confounding factors in this context include miscoding of influenza deaths, income, socioeconomic status, race, age, industrial composition, population density, air pollution, air conditioning usage, school characteristics, and school attendance (32).

Time-period fixed effects \( \Phi_t \) are included to minimize omitted variable concerns that might arise from potential confounders that are common to all sample counties but vary over time. Examples of time-varying confounding factors in this context include macroeconomic shocks that influence health behaviors, technological advances in health treatments, and trends in vaccination rates. Time-period fixed effects are equivalent to fully flexible national time trends.

As an interpretation exercise, we also predicted how much of the difference in influenza mortality between January and July could be explained by seasonal differences in humidity. To do so, we predicted each month’s average influenza mortality as a function of average absolute humidity in that month by multiplying the average frequency of days at a given point on the humidity distribution and our core estimates of the humidity-influenza relation. Second, we calculated the difference between the predicted influenza mortality in January and the predicted influenza mortality in July. Third, we calculated the difference between actual influenza mortality in January and actual influenza mortality in July. Fourth, we divided the predicted difference (step 2) by the observed difference (step 3) to obtain a ratio.

Analysis was carried out with STATA 11 (35). Standard errors used to calculate confidence intervals were clustered to allow for arbitrary serial correlation at the state level.

**RESULTS**

Table 1 summarizes the sample data, over all months and by calendar month. For our 359 urban US counties, monthly influenza mortality rates averaged 0.071. Mortality rates were substantially higher when deaths containing pneumonia as a primary or secondary cause were also included. Influenza mortality was as much as 40 times higher during the cooler months (December to March) than during the warmer months (June to September).

Perhaps the most striking feature of Table 1 is the seasonal correlation between absolute humidity and temperature. The absolute humidity data in column 2 and the temperature data in column 3 have a correlation of 0.96. This large correlation implies that 1) causal relations are unlikely to be correctly identified in models that do not analyze humidity and temperature simultaneously and 2) data sets with a large degree of spatial and temporal variation, like the one used here, are necessary to statistically distinguish the effects of humidity from temperature. Figure 1 illustrates the average cross-state differences in influenza
mortality rates for the years 1973–2002. Like Table 1, the raw averages in Figure 1 are suggestive of a relation between weather and influenza mortality.

Figure 2 shows the main results. The general relation between absolute humidity and influenza mortality follows a downward-sloping exponential shape. Low humidity levels are associated with statistically significant increases in influenza mortality. For example, on average, a shift in the annual distribution of humidity levels that produces 1 additional day at 3 g/kg and 1 less day at 9 g/kg is associated with a 0.6% (95% confidence interval (CI): 0.3, 0.9) increase in mortality relative to the average annual influenza mortality rates for the years 1973–2002. Like Table 1, the raw averages in Figure 1 are suggestive of a relation between weather and influenza mortality.

<table>
<thead>
<tr>
<th>Month</th>
<th>Daily Specific Humidity, g/kg</th>
<th>Daily Temperature, °F (°C)</th>
<th>Daily Precipitation, inches (mm)</th>
<th>Monthly No. of Influenza Deaths per 100,000a,c</th>
<th>Monthly No. of Influenza/Pneumonia Deaths per 100,000b,c,d</th>
</tr>
</thead>
<tbody>
<tr>
<td>All months</td>
<td>7.75</td>
<td>57.7 (14.3)</td>
<td>0.102 (2.59)</td>
<td>0.071</td>
<td>6.254</td>
</tr>
<tr>
<td>January</td>
<td>4.01</td>
<td>38.4 (3.6)</td>
<td>0.102 (2.59)</td>
<td>0.251</td>
<td>8.744</td>
</tr>
<tr>
<td>February</td>
<td>4.30</td>
<td>41.6 (5.3)</td>
<td>0.097 (2.47)</td>
<td>0.242</td>
<td>7.526</td>
</tr>
<tr>
<td>March</td>
<td>5.13</td>
<td>48.3 (9.0)</td>
<td>0.113 (2.87)</td>
<td>0.162</td>
<td>7.419</td>
</tr>
<tr>
<td>April</td>
<td>6.30</td>
<td>56.4 (13.5)</td>
<td>0.104 (2.63)</td>
<td>0.043</td>
<td>6.199</td>
</tr>
<tr>
<td>May</td>
<td>8.58</td>
<td>64.6 (18.1)</td>
<td>0.109 (2.78)</td>
<td>0.014</td>
<td>5.694</td>
</tr>
<tr>
<td>June</td>
<td>11.00</td>
<td>71.9 (22.2)</td>
<td>0.108 (2.75)</td>
<td>0.008</td>
<td>5.221</td>
</tr>
<tr>
<td>July</td>
<td>12.64</td>
<td>75.9 (24.4)</td>
<td>0.104 (2.63)</td>
<td>0.007</td>
<td>5.236</td>
</tr>
<tr>
<td>August</td>
<td>12.61</td>
<td>74.9 (23.8)</td>
<td>0.106 (2.69)</td>
<td>0.006</td>
<td>5.117</td>
</tr>
<tr>
<td>September</td>
<td>10.66</td>
<td>69.2 (20.6)</td>
<td>0.107 (2.71)</td>
<td>0.008</td>
<td>5.103</td>
</tr>
<tr>
<td>October</td>
<td>7.68</td>
<td>59.6 (15.3)</td>
<td>0.087 (2.22)</td>
<td>0.016</td>
<td>5.804</td>
</tr>
<tr>
<td>November</td>
<td>5.72</td>
<td>50.1 (10.1)</td>
<td>0.097 (2.47)</td>
<td>0.022</td>
<td>5.881</td>
</tr>
<tr>
<td>December</td>
<td>4.37</td>
<td>41.5 (5.3)</td>
<td>0.091 (2.31)</td>
<td>0.072</td>
<td>7.108</td>
</tr>
</tbody>
</table>


a All summary statistics were calculated using population weights from 2000.
b Codes from the Eighth Revision of the ICD (ICD-8) were used for the years 1973–1978, codes from the Ninth Revision (ICD-9) were used for the years 1979–1998, and codes from the Tenth Revision (ICD-10) were used for the years 1999–2002.
c Influenza deaths include all deaths for which influenza was listed as a primary or secondary cause of death. The codes for influenza were 470–474 (ICD-8), 487 (ICD-9), and J-10 through J-11 (ICD-10).
d Influenza/pneumonia deaths include all deaths for which influenza and/or pneumonia was listed as a primary or secondary cause of death. The codes for pneumonia were 480–486 (ICD-8), 480–486 (ICD-9), and J-12 through J-18 (ICD-10).

Figure 1. Average annual number of US deaths coded as influenza-related per 100,000 inhabitants, by state, 1973–2002. Shading is based on quartiles, with the lightest gray shade representing the first quartile, the next-lightest gray representing the second quartile, etc. These statistics reflect the authors’ calculations and represent raw averages only; sociodemographic factors and other confounders could potentially drive the observed differences across states. These averages were not adjusted for influenza coding differences across states.
mortality rate. For mean daily specific humidity levels below 6 g/kg, the lower the humidity level the greater the increase in average influenza mortality. Similarly, on average, a shift in the annual distribution of daily humidity levels that produces 1 additional day at 1 g/kg but 1 less day at 9 g/kg is associated with a 1.2% (95% CI: 0.6, 1.8) increase relative to the average annual influenza mortality rate. While there is a statistically significant negative relation between humidity and influenza mortality at mean daily humidity levels below 6 g/kg, this study detected no relation between humidity and influenza mortality at mean daily humidity levels above 6 g/kg.

The detected association between absolute humidity and influenza mortality is practically large, and our results indicate that changes in absolute humidity alone can explain much of influenza’s seasonality in the United States. The core model predicts that approximately one-half (51%) of the difference between the average US January influenza mortality rate and the average US July influenza mortality rate can be explained by average seasonal differences in absolute humidity alone.

The general relation between temperature and influenza mortality followed a bell-shaped curve. We detected no statistically significant relation between temperature and influenza mortality at mean daily temperatures below approximately 15°F (−9.4°C) or above approximately 60°F (15.6°C). Mean daily temperatures between 15°F (−9.4°C) and 30°F (−1.1°C) also were not associated with statistically significant changes in influenza mortality, but point estimates were large in magnitude. Mean daily temperatures between 30°F (−1.1°C) and 60°F (15.6°C) were associated with statistically significant increases in influenza mortality, with the peak influenza mortality impact around a mean daily temperature of 30°F (−1.1°C). On average, a shift in the annual temperature distribution that produced 1 additional day at 30°F (−1.1°C) but 1 less day at 65°F (18.3°C) was associated with a 0.8% (95% CI: 0.1, 1.8) increase in mortality relative to the average annual influenza mortality rate. A shift in the annual temperature distribution that produced 1 additional day at 50°F (10°C) but 1 less day at 65°F (18.3°C) was associated with a smaller, but still significant, 0.3% (95% CI: 0.1, 0.5) increase relative to the average annual influenza mortality rate.

**Sensitivity analysis**

A natural concern is that influenza deaths are not always corroborated virologically. Therefore, following the approach of Dushoff et al. (6), we conducted a sensitivity investigation that replicated the analysis using outcome measures that included both influenza and pneumonia as primary or secondary causes of death. Results, shown in Figure 3, indicated that the qualitative humidity-mortality and temperature-mortality relations were similar to the main results in Figure 2. With much higher death rates from pneumonia and some pneumonia deaths being unrelated to influenza, relative magnitudes were considerably smaller, as expected.

Another concern is that ICD coding practices change over time. To address possible concerns about long-run changes in classification systems, we conducted a sensitivity investigation that replicated the analysis using only the ICD-9 years (1979–1998). Results, shown in Figure 4, indicate that the humidity-mortality relations are similar to the main results in Figure 2. However, the relation between temperature and influenza mortality was less robust. To address possible concerns about seasonal differences in coding, we conducted a sensitivity investigation that replicated the analysis omitting summer months (where the probability of influenza misclassification may be higher). Results, shown in Figure 5, were similar to our main results in Figure 2.
Next, we address concerns that our results are driven by our choice of lag structure (i.e., 2 months). Results in Web Figure 1 (available at http://aje.oxfordjournals.org/) indicate that different exposure lag structures generate qualitative humidity-mortality and temperature-mortality relations that are similar to the main results in Figure 2. The only major difference is that the magnitude of weather-mortality relations is diminished when exposure variables are based on contemporaneous months alone. This suggests that short-term exposure variables may underestimate relations between weather and influenza mortality.

We verify that our estimates are robust to changes in how we control for potential confounders. Results in Web Figure 2 indicate that different fixed-effect approaches generate humidity-mortality relations that are similar to the main results in Figure 2. However, the relation between temperature and influenza mortality is less robust. Note that all specifications with county fixed effects (as opposed to county-by-calendar month fixed effects) generate smaller weather-mortality relations. This suggests that failure to account for local seasonality may cause one to underestimate relations between weather and influenza mortality.

Finally, we investigated whether estimated relations varied geographically within the United States. Unfortunately, a full regional investigation was beyond the scope of our study.

Figure 3. Regression estimates of the relation between influenza/pneumonia mortality and weather conditions in 359 urban US counties during the years in which the International Classification of Diseases, Ninth Revision, was in use (1979–1998). In part A, the solid line depicts the average percentage change in the annual influenza/pneumonia mortality rate from 1 additional day at a given humidity level, relative to 1 additional day with a humidity of 9 g/kg. In part B, the solid line depicts the average percentage change in the annual influenza/pneumonia mortality rate from 1 additional day at a given temperature, relative to 1 additional day with a temperature of 65°F (18.3°C). Dashed lines represent 95% confidence intervals. Point estimates are qualitatively similar to those in Figure 2.

Figure 4. Regression estimates of the relation between influenza mortality and weather conditions in 359 urban US counties during only the years in which the International Classification of Diseases, Ninth Revision, was in use (1979–1998). In part A, the solid line depicts the average percentage change in the annual influenza mortality rate from 1 additional day at a given humidity level, relative to 1 additional day with a humidity of 9 g/kg. In part B, the solid line depicts the average percentage change in the annual influenza mortality rate from 1 additional day at a given temperature, relative to 1 additional day with a temperature of 65°F (18.3°C). Dashed lines represent 95% confidence intervals. In part A, the estimated relation is qualitatively similar to that in Figure 2. In part B, there is no significant relation between temperature and mortality, unlike Figure 2.
DISCUSSION

This study provides novel ecologic evidence that absolute humidity and temperature affect influenza mortality in human populations. Most significantly, this research supports the emerging hypothesis that absolute humidity is a critical determinant of observed influenza outcomes, even after controlling for temperature. These results bolster recent laboratory findings from guinea pigs and state-level epidemiologic evidence (21, 23). An additional key result is that the humidity-influenza relation is nonlinear. Lower humidity levels only result in greater influenza mortality at mean daily specific humidity levels below 6 g/kg. Incremental changes in humidity do not significantly affect influenza mortality when mean daily specific humidity exceeds a 6-g/kg threshold. Temperature appears to be an important determinant of human influenza outcomes, even after controlling for absolute humidity. However, temperature-mortality estimates are sensitive to model specification.

Our results have important implications for public health and policy. Estimates can be used to help predict the location and timing of future influenza mortality. In addition, highlighted nonlinearities between influenza mortality and absolute humidity may be especially useful for understanding regional variation in influenza outcomes triggered by climate change.

The present study’s data and methods permitted an unusually large-scale and robust quantitative assessment of the relation between weather conditions and influenza mortality. However, we note two limitations related to outcome measures. First, the outcome measure (mortality) did not directly incorporate effects on influenza morbidity, which may be more directly linked to humidity and temperature than influenza mortality. Second, all outcome measures are measured with error. Our goal in the present study was to explore the relation between weather conditions and observed influenza mortality, not to explore the total number of deaths that are attributable to influenza. Under the assumption of uncorrelated measurement errors, quantitative assessments of weather-influenza relations from regression models are correct, on average (39). However, regression standard errors may be inflated. As a consequence, it may be more difficult to reject the null hypothesis of no relation between weather conditions (i.e., temperature) and influenza mortality.

We note other limitations. First, we cannot completely rule out confounding factors. Our modeling strategy controlled for confounding factors that are approximately fixed at the county level, vary by season at the county level, or vary across time at the national level. However, omitted variables can bias estimated relations if the omitted factors are correlated with anomalous, rather than typical, weather outcomes. Specifically, pollution concentrations, air conditioning usage, school attendance, and ICD coding errors that vary with unusual weather conditions could have biased our results. Second, our study remains agnostic with regard to specific mechanisms. We cannot definitively differentiate between host susceptibility, disease transmission, and virus survival channels, nor can we definitively identify whether indoor or outdoor exposure drives our results. It is

Figure 5. Regression estimates of the relation between influenza mortality and weather conditions in 359 urban US counties, excluding the months June through September, 1973–2002. In part A, the solid line depicts the average percentage change in the annual influenza mortality rate from 1 additional day at a given humidity level, relative to 1 additional day with a humidity of 9 g/kg. In part B, the solid line depicts the average percentage change in the annual influenza mortality rate from 1 additional day at a given temperature, relative to 1 additional day with a temperature of 65°F (18.3°C). Dashed lines represent 95% confidence intervals. Point estimates are qualitatively similar to those in Figure 2.
interesting to note, however, that the present study’s results show that absolute humidity significantly affects influenza outcomes even after controlling for temperature and precipitation events that may cause crowding indoors. It is unlikely that behavior responds directly to absolute humidity in isolation, so results may be suggestive of virus survival or host susceptibility mechanisms. Third, susceptible–infected–recovered dynamics were not a feature of our model. Fourth, our study was set in the temperate climate of the United States. Our results do not shed light on the long-standing puzzle of rainy-season influenza epidemics in tropical countries or in semitropical regions within the United States (like the Gulf states) (7).

The collective limitations of this study and related research indicate that much work remains. More laboratory and modeling evidence is necessary to better understand influenza mechanisms. In addition, more observational and ecologic evidence is necessary to understand influenza outcomes in a world complicated by human behavior. Nevertheless, this research demonstrates that future explorations that target the role of absolute humidity in influenza mortality may produce high returns.

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